I congratulate the authors again on a most thought-provoking study. I look forward to their feedback about these considerations.

John G. T. Augoustides, MD, FASE
Department of Anesthesiology and Critical Care
Hospital of the University of Pennsylvania
Philadelphia, PA 19104-4283

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Reply to the Editor:

We appreciate the comments provided by Dr Augoustides in regard to our recent article "Ventilatory Dependency After Cardiovascular Surgery." Our article attempts to define the risks for ventilator dependency and determine the outcomes of patients who develop this problem after their index cardiovascular surgery. Two inquiries were made (as points [a] and [c] are closely related).

We were surprised ourselves to find that there has been a continuing trend toward a lower prevalence of ventilator dependency at our institute. A declining trend was noted in previous studies from our institute published several years before. That this trend has continued through the current study has both thrilled and perplexed us. It was impossible for us to determine which quantifiable variables might be responsible for this trend because of a variety of confounding factors, not the least of which is time itself. What is even more interesting is that this phenomenon has occurred in the setting of increasing complexity and acuity of illness. It is our suspicion that several coincident changes might be responsible. There has been a steady improvement in myocardial protection over the years and a progressive increase of intraoperative echocardiography use; these factors likely promote more rapid myocardial recovery. Less narcotics are being given intraoperatively, and this may facilitate earlier separation from mechanical ventilatory support. In addition, there has been a noticeable increase in intensivist staffing, such that there is seemingly more planning and continuity of care in the intensive care unit, which again, might favorably affect weaning.

Second, aprotinin is used sparingly at our institution (in $<1\%$ of patients). This is, in part, secondary to cost, but also for concerns regarding thrombotic complication and renal dysfunction, all without clearly demonstrable benefit. There are 2 populations for whom we do use aprotinin on more occasions: patients with ventricular-assist devices and patients undergoing a second cardiac transplant. These groups were excluded from our study.

Sudish C. Murthy, MD, PhD
Thoracic and Cardiovascular Surgery
The Cleveland Clinic
Cleveland, Ohio

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Prosthesis–patient mismatch after mitral valve replacement: Back to reality

To the Editor:

We read with interest the article of Totaro and Argano.[1] We have, however, several concerns with regard to the validity of the results and conclusions presented in this article. First, the title of the article, "Patient–Prosthesis Mismatch after Mitral Valve Replacement: Myth or Reality?" is inappropriate. Mitral prosthesis–patient mismatch (PPM) is equivalent to a residual mitral stenosis, related to the fact that most prosthetic valves have a hemodynamic performance, and thus a valve effective orifice area (EOA), that is inferior to that of the normal native valve. Thus concluding that PPM is a myth would be equivalent to saying that mitral stenosis (or aortic stenosis for aortic PPM) does not exist, and that this is a benign phenomenon, which is of course not the case. In this regard, several studies have demonstrated that PPM is a frequent hemodynamic phenomenon after mitral or aortic valve replacement.[2-5] The important question is rather to determine the impact of PPM on the hemodynamic, functional, and clinical outcomes, and at which degree of severity and in which categories of patients this impact of PPM becomes statistically significant and clinically relevant. Unfortunately, the data provided by Totaro and Argano[1] do not permit an answer to these important questions.

There are serious concerns about the validity of the Doppler echocardiographic data, and especially of those of the valve EOA. This is a crucial aspect, because the identification and quantification of PPM are based on these data. First, it is intriguing to see that the EOAs measured in vivo by Doppler echocardiography, especially for the 25- and 27-mm valves, were larger than the EOAs measured in vitro by the manufacturer. A recent study has indeed demonstrated that, as opposed to the observation in this study, the in vitro EOAs provided by the manufacturer grossly overestimate the in vivo EOAs and are thus not valid for prediction of PPM.[6] Totaro and Argano also used the label prosthesis size as a surrogate for PPM, whereas previous studies have shown that this parameter is not valid for identification of PPM and prediction of its hemodynamic and clinical consequences.[3,6,7]

Moreover, the huge variability in the EOA measurements for a given prosthesis size (from 1.0 to 4.9 cm^2 for the 29-mm prostheses!), the complete absence of correlation between the EOA and the transprosthetic gradient, and the recording of high transprosthetic gradients (>15 mm Hg) in several patients despite the calculation of large EOAs and indexed EOAs (see Figure 3 of the article[1]) further support the concerns regarding the validity of the EOA measurements and thus the identification of PPM. The presence of gradients greater than 15 mm Hg in the mitral position definitely cannot be considered as "favorable hemodynamics," as concluded by Totaro and Argano.[1] In fine, these observations suggest that a large proportion of the patients included in this series were misclassified with respect to the presence or absence of PPM.

Totaro and Argano only measured the valve hemodynamics and systolic pulmonary arterial pressure at predischarge examination or at 30 days. It is well known that the measurements of valve EOA, transvalvular gradients, and pulmonary pressure in the early postoperative period are often unreliable because of the poor acoustic window, hyperdynamic state, or flow acceleration in the left ventricular outflow tract.